Every reasonable effort should be made to identify the etiologic agent so that an appropriate therapeutic program can be initiated. Since the previously high mortality of tuberculous pericarditis has been remarkably reduced by the early administration of an appropriate drug regimen, this practice is recommended whenever tuberculous pericarditis is suspected. It is important to establish the diagnosis of a specific fungal pericarditis because of differences in antimicrobial chemotherapy. The indications for pericardiocentesis, corticosteroids, surgical drainage and pericardiectomy in the management of this form of pericarditis have been reviewed.

Bacterial Pericarditis

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THE CLINICAL signs and symptoms of bacterial (purulent) pericarditis are similar to those of the other forms of pericarditis except that the patients are usually more toxic and ill with rapidly changing hemodynamic abnormalities.

Bacterial pericarditis is an old disease first described in an animal by Galen in the first century and subsequently described in humans approximately a thousand years ago. The incidence of bacterial pericarditis from autopsy series before the antibiotic era was about 40 percent of all pericarditis;[57] subsequent to the antibiotic era the incidence has decreased to 10 to 20 percent.[57] Additionally, autopsy series showed that purulent pericarditis before 1945 was responsible for approximately 1 percent of all deaths.[57] Since 1945, however, the figure has been less than 1 percent.

The pathogenesis of bacterial pericarditis occurs through several mechanisms:

- *Contiguous spread from an adjacent pleuropulmonary infection.* Before the antibiotic era, most cases of purulent pericarditis were due to the pneumococci and this was invariably associated with pneumonia or empyema or both. The postulated pathophysiology has been that the adjacent pleuropulmonary infection may cause an inflammatory response in the pericardium with migration of neutrophils and eventual deposition of fibrin.[26,58,59] It has not been clear whether bacteria migrate directly from the lung tissue itself or there is subsequent bacteremia and invasion of the pericardial sac.

- *The hematogenous spread of bacteria from a distant focus to the pericardium.* Staphylococcal osteomyelitis is a common cause of purulent pericarditis and the pathogenesis is probably via hematogenous spread of the bacteria to pericardium. The exact mechanism is unknown. Animal experiments[60] suggest that an initial myocarditis or myocardial abscess develops. Bacteria then migrate (or rupture from the abscess) into the pericardium to form purulent pericarditis.

- *Direct inoculation of bacteria into the pericardial sac.* This may result from penetrating wounds to the pericardium or occur as a result of cardiac surgery.[57] There have been reports of bacterial pericarditis secondary to infective endocarditis.[61-63] Associated myocarditis with myocardial abscess formation probably permit migration of bacteria to the pericardial space.

The incidence of particular types of bacteria in purulent pericarditis is difficult to assess since most series include only a few case reports. However, a review of the world's literature up to 1959[26] of all cases of purulent pericarditis suggested staphylococci, pneumococci and streptococci as the predominant organisms. There were a moderate number of meningococci reported; since that report, the incidence has increased. In children under the age of two years, the cause of purulent pericarditis is similar to that in adults with staphylococci and pneumococci predominating, but Hemophilus influenza is also a common agent.[64] Hemophilus influenza should always be considered in the pediatric age group. Salmonella species may also cause bacterial pericarditis.[65] Most have been nontyphosal types with Salmonella typhimurium being the most common strain.

Pneumococcal pericarditis was the most common cause of purulent pericarditis before the antibiotic era. However, a recent review by Kauffman and coworkers[59] indicates that there have only been 15 cases of pneumococcal pericarditis reported since 1945. This is probably a reflection of penicillin therapy. Kauffman's group included five cases of their own in this report and it is in-

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teresting that two of their patients had a history of chronic alcoholism and one patient had hypogammaglobulinemia. The three patients that did recover were diagnosed antemortem and the two who died were diagnosed postmortem. Most series indicate that the antemortem clinical diagnosis of bacterial pericarditis occurs 10 to 20 percent of the time, and before the antibiotic era the mortality was essentially 100 percent. In the same report by Kauffman's group, the cumulated cases of pneumococcal pericarditis from other series again showed the high association with pneumonia and empyema. Therefore, pericarditis should be considered a possible complication in patients with pneumococcal pneumonia and empyema.

In staphylococcal pericarditis, hematogenous spread from a distant foci is very common except in children where staphylococcal pericarditis is more commonly associated with pulmonary infection. Osteomyelitis and peripheral skin infections have been the most common sources of bacteremia. In terms of mortality, staphylococcal pericarditis has the worst prognosis.

Meningococcal pericarditis appears to be increasing in frequency despite the availability of antibiotics.[66] Meningococcal pericarditis is almost always associated with meningitis or meningococcemia or both. To my knowledge, there have been only seven reported cases of nonmeningitic meningococcal pericarditis reported in the literature.* Two different pathogenic mechanisms may be involved in meningococcal pericarditis. One type of pericarditis is seen early in meningococcal infections when the patient is acutely ill and probably results from direct bacterial invasion of pericardium or hematogenous spread with an associated myocarditis. There is also a late form of meningococcal pericarditis in which pericarditis develops a week or more after antibiotic therapy. The pathogenesis may be either an immunologic or nonimmunologic response to meningococcal endotoxin released from bacterial cells.* Fever, pleural effusion, arthritis or arthralgia are seen in many of these patients. The pericardial fluid and other lesions have been cultured with no bacteria recovered. It is important to remember this syndrome because most of these patients will respond to anti-inflammatory agents such as acetylsalicylic acid or steroids and needle aspiration.

As far as complications of bacterial pericarditis are concerned, it is important to emphasize that cardiac tamponade is the cause of death for most patients with this disease mainly because of failure in making an early diagnosis. Congestive heart failure from associated myocarditis may be a major problem. Constrictive pericarditis is relatively uncommon in bacterial pericarditis.[26,57,64] In patients with purulent pericarditis who have been followed clinically, constrictive pericarditis has failed to develop, as opposed to patients who have tuberculous pericarditis—in whom this complication not uncommonly develops.

In the management of bacterial pericarditis, the single most important factor is a high index of suspicion. Only 10 to 20 percent of these cases are diagnosed antemortem. Because one of the pathogenic mechanisms is bacteremia, peripheral foci of infection must be looked for and appropriate cultures obtained including blood. Evidence for associated pleuropulmonary infection should be investigated and appropriate cultures taken. Careful cardiac and hemodynamic evaluation should be made. Pericardiocentesis should be carried out in suspected cases of purulent pericarditis. The etiological diagnosis will not be made unless some pericardial fluid is examined. Once the fluid is obtained, Gram and acid-fast stains should be made immediately and the fluid cultured for bacteria, fungi, virus and Myobacterium tuberculosis. The characteristics of the aspirated fluid should be noted and examinations for cell count, differential, glucose and protein obtained.

The antibiotics most frequently used until the cause is established are penicillin or methicillin or both, administered intravenously in large doses. In the pediatric age group, ampicillin instead of penicillin should be used if Hemophilus influenza is thought to be the cause.

Drainage is extremely important. Repeated pericardiocentesis using a percutaneous approach should be used if reaccumulation occurs. If adequate drainage cannot be achieved, then an open surgical drainage should be instituted. If, beyond that, drainage becomes a problem because of tamponade, pericardiectomy may be indicated. With the institution of systemic antibiotics and adequate drainage, survival rates of 50 to 75 percent can be achieved.[26,57] However, improvement on these figures can only be achieved with early recognition of bacterial pericarditis and prompt initiation of therapy.

*Unpublished data submitted for publication.